Fasting proinsulin level is not associated with first phase insulin response in impaired glucose tolerance

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published in
Diabetes
1997

document version
Publisher's PDF, also known as Version of record

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Fasting Proinsulin Level is not Associated with First Phase Insulin Response in Impaired Glucose Tolerance.

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High fasting proinsulin levels (FPI) are suggested to reflect *B*-cell dysfunction, and have been shown to predict the conversion from impaired glucose tolerance (IGT) to diabetes. The aim of this study was to assess the relationship between FPI and *B*-cell function as measured with the hyperglycemic clamp (10 mmol/l). *B*-cell function was measured in 96 subjects, aged 45-74 yrs, with IGT (based on the mean of 2 oral glucose tolerance tests) as the first phase (0-10 min) delta area under the curve of specific insulin, having a median and interquartile range of 615 (254-1128) pmol/l/min. The table depicts the fasting plasma glucose (FPG), fasting proinsulin (FPI) and fasting ratio proinsulin/insulin for the lowest and highest quartiles of the first phase insulin response.

First phase ins response	FPG (mmol/l)	FPI (pmol/l)	Ratio FPI/I
<254 (pmol/l/m) n=24	6.4 (0.7)	4.5 (2.7)	0.08 (0.04)
>1128 (pmol/l/m) n=24	5.8 (0.9)*	6.0 (7.0)	0.07 (0.06)

*) $p<0.05$ vs. lowest quartile. Data are non-adjusted mean values (SD). A multiple linear regression analysis was performed using the first phase insulin response as dependent variable, and fasting proinsulin as independent variable, adjusted for insulin sensitivity (glucose infusion rate/mean insulin level during the last 30 min of clamp). The unadjusted and adjusted regression coefficient (95% confidence intervals; R-square) were 0.1 (-0.2 - 0.4; 0%) and -0.2 (-0.4 - 0.1; 1%). We therefore conclude that fasting hyperproinsulinemia does not reflect a defective first phase insulin response in subjects with IGT.

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A U-Shaped Relationship Between Waist Circumference And Gestational Glucose Intolerance In Brazilian Women.

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We have previously reported a positive relationship between central fat distribution and gestational glucose tolerance in approximately 1000 generally well-nourished women in Southern Brazil. In order to examine this association in greater detail, we investigated the relationship of waist circumference with gestational glucose intolerance (GGI), defined as glycemia ≥ 140 mg/dL 2h after a standard 75g glucose load, in pregnant women aged 20 years or more, without known diabetes outside pregnancy, enrolled consecutively in 6 Brazilian capital cities. Data pertain to the 4929 women with complete information. Odds of presenting GGI by quartile of waist circumference were estimated through logistic regression analyses controlling for enrollment center, age, sum of skinfold thicknesses, height, family history of diabetes, previous gestational diabetes, skin color, patient referral pattern, and gestational age:

Waist Circumference (quartile)	Odds Ratio (OR) total sample	non-white women	short women
1st	1.54*	1.96*	1.83*
2nd (reference)	1.00	1.00	1.00
3rd	1.17	1.24	1.16
4th	1.69*	2.27*	2.02*

*p < 0.05

The unantecipated, statistically significant, U-shaped relationship shown above, present in the overall sample, was accentuated in non-white women and those of shorter stature. These data confirm that central obesity associates with gestational glycemia. They suggest, however, that central leanness, at least in our pregnant population, many of whom have suffered chronic malnutrition, is also associated with increased GGI.

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Weight Change Modifies Associations Between Nutrient Intake and lipids in NIDDM: The Insulin Resistance Atherosclerosis Study (IRAS).

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Dietary guidelines for persons with NIDDM caution against high intake of carbohydrate (CHO) which may exacerbate dyslipidemia, particularly when weight loss is unsuccessful. Among 438 persons with NIDDM by WHO criteria who participated in the IRAS exam, self-reported weight change in the last year was: 21% gained 5+ pounds, 31% lost 5+ pounds, and 48% maintained stable weight. Usual diet over the last year was assessed by a food frequency interview that incorporated ethnic-specific foods. Regression models were done separately for each lipid (outcome) and each nutrient (exposure), adjusted for total calories, baseline body mass index, physical activity, alcohol intake, diabetes medication and duration and demographic variables. Results as follows are given according to weight change status from models in which the (weight change x nutrient) interaction term had a p-value <0.10.

Among those who gained weight, higher intakes of total, simple, and complex CHO were associated with higher triglycerides (TG) (each $p<0.01$); total and saturated dietary fats were positively but not significantly related to LDL-cholesterol (each $p>0.10$). Among those who lost weight, CHO intakes were not related to TG (each $p>0.10$) but higher intakes of total and saturated fats were associated with higher LDL-C (each $p<0.02$). Among those with stable weight, CHO intakes were not related to TG (each $p>0.10$) and higher intake of total fat was related to higher LDL-C ($p<0.05$). Thus, weight change (independent of baseline weight) may be an important determinant of the impact of habitual diet on lipoprotein profile in community-dwelling NIDDM patients.

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Development of Proliferative Diabetic Retinopathy in African Americans and Whites with Type 1 Diabetes.

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African Americans are at a higher risk of developing microvascular complications of diabetes. Our preliminary study had shown no difference between African Americans and whites with type I diabetes in the development or progression of diabetic retinopathy until adjustments were made for the worse risk factor profile. In the current larger study (n=312) covering a longer time span (mean of 8.4 yr), we examined racial differences in the development of proliferative diabetic retinopathy (PDR). All subjects had type 1 diabetes and at least 2 sets of gradeable eye photos (stereo, color, 7 standard fields). Excluded were subjects with pre-existing PDR, laser treatment or hemoglobinopathy. Masked grading was conducted at the Wisconsin Reading Center. All subjects were assessed using a standard protocol. At baseline African Americans (n=97) had worse glycemic control (mean HbA1 of 11.3 vs 10.0%; p<.0001), higher systolic blood pressure (mean of 117 vs 110 mmHg; p<.001) and were older (mean of 26.8 vs 19.3 yr; p<.0001) than the white subjects. There were no differences in duration of diabetes or length of follow-up by race. In the African Americans, 17.5% developed PDR compared to 10.2% in the 215 whites for an odds ratio of 1.86 (95% CI 0.94-3.70). When adjusting for baseline glycemic control, retinopathy grade, and length of follow-up, race was still not a significant risk factor with the adjusted odds ratio for race reduced to 0.73 (95% CI .30-1.78). In race-specific models, the odds ratio for glycemic control was non-significantly higher in whites (OR=2.41 95% CI 1.42-4.08 per change of 2% HbA1) compared to that for African Americans (OR=1.66 95% CI 0.97-2.83). We conclude the higher incidence of PDR in African Americans appears to be explained by the worse risk factor profile.

ADA Funded Research

A numeral beside an author's name indicates a duality of interest. See Duality of Interest Information beginning on page lxxxvii.